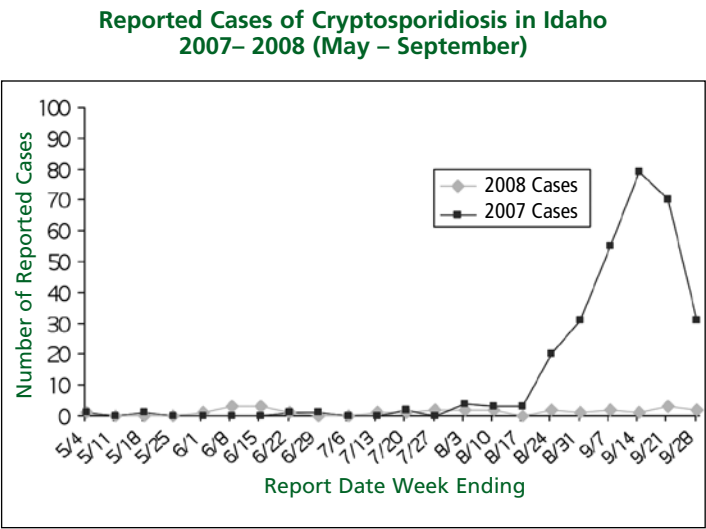


Cryptosporidiosis Data Snapshot

CRYPTOSPORIDIOSIS, A WATERBORNE ILLNESS caused by the chlorine-resistant parasite *Cryptosporidium*, has been reportable in Idaho since 2000. From 2000–2006, 15 to 40 cases were reported annually. However, in 2007, Idaho had 515 reported cases of cryptosporidiosis, over 13 times more than any other year. The reasons for this outbreak remain unclear. Possible explanations include a real increase in disease transmission, enhanced surveillance, and an increase in healthcare provider testing for *Cryptosporidium* following the recent approval of nitazoxanide for treatment of cryptosporidiosis in children and adults. Presumably, a combination of these factors, perhaps influenced by the hot and dry conditions last summer, caused the dramatic increase. In 2008, significantly fewer cases of cryptosporidiosis have been reported. The reason for the precipitous drop in reported cryptosporidiosis cases is unclear. Conceivably, public education regarding safe swimming practices, along with improved sanitation facilities at area splash parks, at least in part, may have led to fewer infections and a subsequent decrease in the number of reported cryptosporidiosis cases. For more information, see <http://www.rwi.dhw.idaho.gov>.



ROUTINE 24-Hour Disease Reporting Line 1.800.632.5927
EMERGENCY 24-Hour Reporting Line 1.800.632.8000

An electronic version of the Rules and Regulations Governing Idaho Reportable Diseases may be found at <http://adm.idaho.gov/adminrules/rules/idapa16/0210.pdf>
Current and past issues are archived online at www.epi.idaho.gov.

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Biosafety Level 3 Laboratory Facility

The Idaho Bureau of Laboratories (IBL) is pleased to announce that the IBL's Biosafety Level 3 (BSL-3) laboratory facility is now operational. The 3,000 sq ft laboratory is a state-of-the-art biocontainment suite that houses the mycobacteriology laboratory and Idaho's only Laboratory Response Network (LRN) reference laboratory. The LRN is a national network of public health laboratories that responds to biological and chemical terrorism, emerging infectious diseases, and other public health emergencies.

Laboratory biosafety levels range from BSL-1, the least restrictive, to BSL-4, for work with agents that pose a very high risk of aerosol-transmitted laboratory-acquired infections and life-threatening disease. A BSL-3 laboratory is designed to contain agents that may cause serious or lethal disease as a result of inhalation. Examples of microorganisms assigned to this biosafety level include *Mycobacterium tuberculosis*, St. Louis encephalitis virus, and *Coxiella burnetii*, the agent that causes Q fever. The emphasis of BSL-3 level facility design and operation is on providing barriers to protect laboratorians and the environment from aerosol exposure and is the appropriate containment level for work with highly pathogenic influenza strains, SARS-CoV, and variola (smallpox) virus screening.

The new facility is comprised of three BSL-3 laboratories, each containing 2 biosafety cabinets, sample storage and receiving area, and support rooms, including one with shower in/out capability and advanced environmental controls. In addition to biosafety equipment and advanced environmental controls, the BSL-3 laboratory is equipped to decontaminate all liquid and solid biowaste before it leaves the facility.

The new laboratory will better serve the Idaho medical and first-responder communities through increased capacity to identify naturally occurring and man-made agents detrimental to human health. During the national anthrax attacks of 2001, IBL received nearly 100 samples from suspected "white powder" incidents over a 6–8 week period. Due to the lack of a BSL-3 laboratory at that time, it was necessary to displace laboratorians from the mycobacteriology laboratory to analyze samples, placing a strain on laboratory resources. IBL continues to receive "white powder" specimens for analysis and potentially hazardous microbiological isolates for confirmatory or rule-out testing. The new BSL-3 facility provides both ample capacity to respond to public health emergencies as well as the ability to more effectively analyze microorganisms such as *Brucella sp.* (brucellosis) and *Burkholderia sp.* (e.g., *melioidosis*) in a safe and secure environment.



IBL microbiologists Walt DeLong and Vivian Lockary cut the ribbon on the new laboratory.

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Infectious Etiologies of Chronic Diseases

AN INCREASING BODY OF RESEARCH is providing evidence of infectious agents as possible etiologies of various chronic conditions. In doing so, this research illustrates the dynamic nature of disease categories such as “infectious” and “chronic.” While many health disciplines commonly regard “infectious” and “chronic” diseases as mutually exclusive entities, closer inspection reveals the distinction to be less clear.

It has been argued that infections are acute events and, therefore, the opposite of chronic. Yet the flaws in this logic are readily apparent as evidenced by many infections (HIV, hepatitis C, herpes simplex) that result in chronic illness. Likewise, heart disease is usually classified as chronic, but is considered acute for the heart attack victim who dies en route to a hospital.¹ A long latency is thought to be a hallmark of chronic diseases; however, some infectious diseases (e.g., TB, herpes zoster, AIDS) have long latency periods. Even transmissibility from person to person does not appear to distinguish between infectious and chronic diseases. Legionnaires’ disease, Valley fever, and foodborne botulism are classified as infectious despite not being propagated from person to person, while

dental caries resulting from bacterial infection are directly transmissible between people yet almost exclusively considered a chronic condition.

Despite our tendency to regard the infectious or chronic disease distinction as concrete, researchers continue to explore linkages that have always existed between infectious and chronic disease. These investigations have seldom demonstrated a straightforward relationship, but have often revealed a complex interplay of pathogens, human evolution, and chronic disease. Indeed microbes are rarely, if ever, seen as the sole etiologic agent of a chronic illness, but rather viewed as “triggers” that set in motion or expedite a disease process.²

Perhaps the most renowned discovery pertaining to the infectious origins of a chronic disease is that of *Helicobacter pylori* and ulcers nearly 26 years ago. So dogmatic was our belief that stress and lifestyle factors were responsible for ulcers that widespread acceptance of the role of *H. pylori* didn’t come about until very recently. Since about 2000, *H. pylori* has been proven to cause gastric tumors and speculated to cause multiple other conditions (Tables 1 and 2). Treatment has been so effective that *H. pylori* is now considered an

“endangered bacterial species” in the developed world.³ Additionally, there is growing speculation that, since *H. pylori* and humans have co-existed for thousands of years, these bacteria provide a protective effect against other chronic conditions. Thus, there is an urgent need to more thoroughly assess risks and benefits of *H. pylori* to settle the debate and determine strategies on how best to address the presence of this bacterium in people.

The expanding list of chronic diseases with suspected infectious etiology reminds us that categorizing a disease is a dynamic process. As research linking chronic conditions to microbial triggers progresses, it becomes increasingly important that clinicians be kept up to date. Clinicians who are aware that pathogens can be involved in more diverse diseases than previously thought have played important roles in bolstering claims of an infectious etiology, as a result of incisive clinical observations. As these claims are strengthened, clinicians will have an equally important role in avoiding potential negative consequences (e.g., increasing risky behavior due to disease being perceived as easily curable) by carefully conveying this information to patients.

Table 1. Chronic Diseases for Which There is Strong Evidence of an Infectious Etiology¹

<i>Chronic Disease</i>	<i>Infection</i>
Adult T cell leukemia	Human T-cell Lymphotropic virus type 1
Tropical spastic paraparesis	
Cervical carcinoma	
Laryngeal papilloma	
Penile cancer	
Anal cancer	Human papilloma virus
Vulvar and vaginal intraepithelial neoplasia	
Venereal warts	
Common warts	
Head and neck cancer	
Burkitt’s lymphoma in Africa	Epstein-Barr virus
Nasopharyngeal carcinoma	
Hodgkin’s disease	
Post-transplant lymphoproliferative disorders	
B cell lymphomas in AIDS patients	
Hepatocellular carcinoma, chronic hepatitis	Hepatitis B virus (HBV)
	Hepatitis C virus (HCV)
	HBV and delta virus
Polyarteritis nodosa	HBV
Mixed cryoglobulinemia	HCV
Sub acute sclerosing panencephalitis	Measles
Multicentric Castleman’s disease	Kaposi’s sarcoma-associated herpes virus
Lymphoma	
Kaposi’s sarcoma	
Anemia; arthritis	Parvovirus B19
Post-rubella arthritis syndrome	Rubella
Congenital rubella syndrome	
Creutzfeldt Jacob disease	Prions
Kuru	
Familial insomnia	
Gastric lymphoma	<i>Helicobacter pylori</i>
MALT lymphoma	
Peptic ulcer disease	Histoplasmosis
Chronic pericarditis	
Lyme disease	<i>Borrelia burgdorferi</i>
Post-streptococcal glomerulonephritis	Group A <i>Streptococcus</i>
Reiter’s syndrome and reactive arthritis	<i>Chlamydia trachomatis</i>
Guillain-Barré syndrome	<i>Campylobacter jejuni</i>
Pelvic inflammatory disease	<i>Chlamydia trachomatis</i>
Squamous cell carcinoma	Osteomyelitis
Hemolytic uremic syndrome	<i>Escherichia coli</i> O157:H7

Table 2. Chronic Diseases for Which There is Suspicion of an Infectious Etiology⁴

<i>Disease</i>	<i>Suspected Agent(s)</i>
Primary biliary cirrhosis	<i>H. pylori</i> , retrovirus
Mesothelioma	Simian virus 40
Multiple sclerosis	Epstein-Barr virus
Tics and obsessive compulsive disorder	Group A <i>Streptococcus agalactiae</i>
Obsessive compulsive disorder	Group A <i>S. agalactiae</i>
Crohn’s disease	<i>Mycobacterium paratuberculosis</i> and others*
Alzheimer’s disease	<i>Chlamydia pneumoniae</i>
Diabetes	Enteroviruses
Sjogren’s disease	<i>H. pylori</i>
Sarcoidosis	<i>Mycobacterium spp.</i>
Atherosclerosis	<i>C. pneumoniae</i> , CMV
Bell’s palsy	Herpes simplex virus
Schizophrenia	Intrauterine exposure to influenza
ALS	Prions
Chronic fatigue	HTLV-1; EBV
Prostate cancer	BK virus

**Clostridium*, *Campylobacter jejuni*, *C. faecalis*, *Listeria monocytogenes*, *Brucella abortus*, *Yersinia pseudotuberculosis*, *Y. enterocolitica*, *Klebsiella spp.*, *Chlamydia spp.*, *Eubacterium spp.*, *Peptostreptococcus spp.*, *Bacteroides fragilis*, *Enterococcus faecalis*, and *Escherichia coli*

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Case Study in Environmental Medicine

WHETHER IT IS RESPIRATORY PROBLEMS resulting from air pollution in Beijing, risk to infants from Bisphenol A in baby bottles, melamine in formula, or lead poisoning from children’s toys, conditions attributable to environmental exposures are increasingly receiving attention in the national media. For physicians these conditions pose diagnostic challenges since many environmental diseases may masquerade as common medical problems or cause nonspecific symptoms. The key to diagnosis is to consider the possibility of environmental factors of disease. By taking a thorough exposure history, primary care physicians can play an important role in detecting, treating, and preventing disease due to toxic environmental exposures.

Consider the following scenario:

“On Tuesday afternoon, a 52-year-old man with previously diagnosed coronary artery disease controlled by nitroglycerin describes episodes of recurring headache for the past three weeks. Mild nausea often accompanies the headache; there is no vomiting. He describes a dull frontal ache that is not relieved by aspirin. The patient states that the headaches are sometimes severe; at other times they are a nagging annoyance. The durations range from half an hour to a full day.

“His visit was also prompted by a mild angina attack that he suffered this past weekend shortly after he awoke on Sunday morning. He has experienced no further cardiac symptoms since that episode.”

Multiple possibilities exist for his headache and nausea. Would you include exposure to toxicants in your differential diagnosis? Are the headaches and cardiac symptoms related? If you learned that the patient refinished furniture as a hobby, worked at a commercial cleaning service, or recently remodeled his home would your differential diagnosis change? Each of these factors could play a role in the etiology of this patient’s illness.

The preceding example was excerpted from a case study developed by the Agency for Toxic Substances and Disease Registry (ATSDR). ATSDR has developed a series of case studies on its website to increase primary care providers’ knowledge of hazardous substances in the environment and to aid in the evaluation of potentially exposed patients. CMEs, CNEs, and other continuing education credits are offered for completing these case studies at <http://www.atsdr.cdc.gov/csem/csem.html>. To learn how the scenario mentioned above can result in this case study patient’s illness, and how to take an exposure history, go to http://www.atsdr.cdc.gov/csem/exphistory/ehcover_page.html!